Idiopathic guttate hypomelanosis	progressive macular hypomelanosis
* 2-5 mm flat white spots found on the shins and forearms (sun exposed parts)	* larger circular areas mostly located on the trunk
* affects fair skinned individuals	* affects dark skinned individuals
* well circumscribed macules with no scaling or atrophy	* poorly defined circular lesion
Cause:-	
* theories:-	theories
* Sun damage ~~since~~	* genetic factor
* Non sun related Seborrhoeic keratosis	* Acne bacteria propionibacter have been found in some cases
* Not preceded by inflammation	* not preceded by inflammation
Diagnosis	
* Skin biopsy	Skin biopsy
① No melanin in skin cells	① ↓↓ melanin
② ↓↓ melanocytes	② normal melanocytes
③ thinner skin	③ ~~Skin~~ No skin thinning
NB Some consider it as apart of aging and needs No ttt	
Treatment to improve appearance:-	Treatment
① light Cryotherapy	① NB-UVB
② dermabrasion	② oral tetracycline
③ Tretinoin cream	③ topical acne preparation
④ Cosmetic Cover up	④ isotretenoin

Progressive macular Hypomelanosis	Idiopathic guttate hypomelanosis.
• It's acquired hypomelanosis may be due to p. Acne in hair follicles.	• It's acquired Leukoderma of unknow cause.
• site: trunk sparing face, Neck.	• site. Sun-exposed are & ē age.
• C/p: hypopigmented macules, nummular, confluent, symmetric ē ill-defined borders.	• C/p: hypopigmented macules, oval, rounded, tear-sha ē sharp borders.
• ↓ melanin pigment.	•
• No scales	• No scales.
• pink fluorescence on wood's light	• No fluorescence.
• tt: - 5% Benzoyl peroxide - 1% clindamycin	• tt: - intralesional injection of triamcinelone @ topic - sun screens - autologus minigrafting.

⑧ D.D of vitiligo

q3

- other Causes of hypopigmentation:

1. Nevus anemicus
2. halo nevus
3. post-inflammatory hypopigmentation/depigment.
4. chemical Leukoderma
5. Nevus depigmentosus (early onset)
6. Leukoderma associated ε scleroderma
7. pityriasis versicolor
8. pityriasis alba
9. Cutaneous manifestation of leprosy, treponematosis (endemic ⊕), onchocerciasis
10. prior ttt ε potent corticosteroids.
11. mycosis fungoides mimicking vitiligo
12. vogt-koyanagi-Harada ⊕
13. idiopathic guttate Hypomelanosis
14. piebaldism
15. IGH, PHH

① Nevus anemicus:

- present at birth
- patch appear paler than the surrounding skin due to vasoconstriction of Blood vessels
- its ill-defined, rubbing surrounding skin → make subtle lesion more prominent

② Halo Nevus:

- Central uniformly Coloured nevus
- Surrounded by peripheral halo of hypopigmentation

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(3) post-inflammatory hypo/depigment (after psoriasis, eczema...
- There is H/o of pre existing dermatoses

(4) piebaldism:
- present at birth
- circumscribed, stable
- white patch
- affecting central face & localized poliosis.

(5) pityriasis versicolor:
- mycologic examination: reveals hyphae, spores.

(6) pityriasis alba.
- seen mainly in children
- scaly hypopigmented patches
- & indistinct border (poorly-defined)
- Commonly on face, Neck

(7) Cutaneous manifestations of leprosy
- anaesthetic disturbance of sense (loss of sensation)

(8) chemical leukoderma.
- It occurs in workers exposed to PSPs (para-substituted phenolic compounds) / Confetti macules.
- No leukotrichia, No Alopecia A.
- white - off white/

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(9) Vogt – Koyanagi – Harada S:

- Aseptic meningitis → initial symptom
- followed by
  - Uveitis – deafness
  - vitiligo – Alopecia areata
  - poliosis of eyelashes / eye brows.

(10) Leukoderma associated sclerodema:

- hardening, sclerosis of skin
- sclerodactyly
- periangual telangectasia
- Calcinosis.

(11) onchocerciasis:

- Severe itching
- blindness
- Called river blindness
- Caused by bite of black fly (beside ri

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Q5 topical tt of melasma"

(I) Daily application of sunscreens ≥ broad spectrum with spf > 30 e.g spectroban, photoderm max.

(II) Bleaching preparation

A- Hydroquinone (HQ) 2-4% e.g Eladoquine 2%

m.o.A → Tyrosinase inhibition
→ Melanocyte DNA & RNA ~~int~~ synthesis inhibition.

Side effects: irritation, transient or permanent depigmentation
- onchronosis
- allergic contact dermatitis
- Carcinogenic in animal studies but not proven in clinical studies

B- Tretenoin gel 0,05% alone or with Hydroquinone.
tretenoin ~~Hydroquinone~~ facilates ~~absorption of~~ penetration of hydroquinone.

M.o.A → Tyrosinase inhibition
→ ↑ Keratinocyte turnover
→ ↑ Keratinocyte pigment granule dispersal.

S/E: Erythema, irritation & desquamation

Page : Date : / /

C Azelic acid (20%) e.g.s

example: Skinoren

m.o.A → Tyrosinase inhibition
→ melanocyte proliferation inhibition
→ Ras suppression
→ Selective effects on hyperactive & abnormal melanocytes

Azelic acid + tretenoin → more effective than azelic acid alone & superior to hydroquine

S/E → irritation, pruritus & erythema

D Kojic acid 2-4%

M.o.A → Tyrosinase inhibition
→ by chelation of Copper essential for tyrosinase

S/E → Irritation & Sensitization

E N-acetyl 4-S cysteaminyl phenol

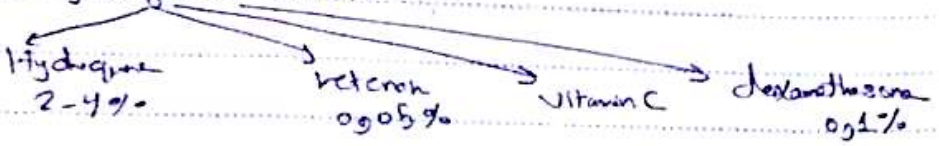
Page : Date : / /

[III] Combined preparations

- Glycolic acid and hydroquinone ~~preparations~~ Combinations.

- Tretenoin containing Combinations :

e.g Kligman's formula



* Vitamin C → Keep the formula stable for long period

Steroid → to reduce irritation caused by tretenoin

[IV] Chemical ~~peel~~ peeling

- M.O.A →

Removal of melanin though thinning of stratum corneum & epidermolysis

S/E → erythema, atrophy, scarring, hypopigmentation, post inflammatory hyperpigmentation

- Example :- TCA (~~used~~ trichloroacetic acid 30-35%)
Jessener's solution
glycolic acid 50-70%.

Types :- superficial peels → remove stratum corneum & enhance penetration of bleaching agent.

Deep peels → remove epidermis & dermis

- post peel hydroquinone, ~~tretenoin~~ pigment or corticosteroids are required for all peels

التاريخ:

موضوع الدرس:

Q 6 Different fitzpatrik skin types.

This classification is based on the person's reaction to Sun exposure

Type I : Always burns , Never tans
Type II : Usually burns than tans.

Type III : May burn , tans well.
Type IV : Rarely burn , tans well

Type V : Rarely burns , tan dark easily
Type VI : Never burn , always tan darkly.

Q.7 Skin Chromophore

- Skin Chromophores are the targets of Selective photothermolysis (i.e Selective destruct of these targets while sparing surrounding structures).
- Important Chromophores in skin include:
  1- Haemoglobin (present in dermal vascular network)
  2- Melanin (found on melanocytes & nearby epi. cells & hair)
  3- Water (Constitute major Component of every cell)
- each Chromophore selectively absorbs light of certain wavelengths "absorption spectrum".
- Selective destruction of certain target necessitate adjustment of 3 laser parameters
  ① wave length ② power ③ pulse duration.

Chromophore	Absorption spectrum	Applications.
① Hb	UV visible near IR	Vascular lesions
② Melanin	UV visible near IR	pigmented lesions & Hair reduction
③ water	Mid-fol IR	- Skin resurfacing - Vaporization of epidermal lesions as (warts)

Adapted mainly from: Bolognia et al., Dermatology Textbook. Third edition. 2012.

Etiologic theories Q8 Pathogenesis of vitiligo ? 1

1. **Autoimmune hypothesis:** This is suggested by:
   - The frequent association with other autoimmune diseases, e.g. thyroid disease (30%), pernicious anemia, Addison's disease, diabetes mellitus, alopecia areata, halo nevus, chronic mucocutaneous candidiasis (CMCC) and uveitis.
   - ***Humoral immunity:*** Autoantibodies are directed against several melanocyte antigens such as:
     - Tyrosinase.
     - Tyrosinase-related proteins 1 (TYRP1).
     - Dopachrome tautomerase.
     - Melanin-concentrating hormone receptor-1 (MCHR1).
     - Transcription factor SOX10.

     The frequent, simultaneous presentation of vitiligo with melanoma is due to the presence of anti-melanoma Abs destroying normal melanocytes in patients with metastatic disease.
   - ***Cellular immunity:***
     - Infiltration of CD8+ T cells into perilesional vitiligo skin.
     - Skin-homing, melanocyte-specific cytotoxic T-cells (e.g. against surface proteins such as Melan-A/MART-1) frequently detectable in the peripheral blood of vitiligo patients.

2. **An intrinsic defect in melanocytes:**

A) • Structural and functional abnormalities of melanocytes:
  - Dilation of the rough endoplasmic reticulum.
  - Abnormal synthesis/processing of TYRP1.
  - Increased sensitivity to oxidative stress (e.g. UVB).

B) • Defective melanocyte adhesion → Detachment and transepidermal loss of melanocytes (melanocytorrhagy).

NB: Induction of detachment by mechanical stress can explain the Koebner phenomenon.

C) • Reduced melanocyte survival and dysregulation of melanocyte apoptosis.

Deficiency of factors important for melanocyte maintenance (e.g. stem cell factor "SCF", microphthalmia-associated transcription factor "MITF").

3. **Melanocyte self-destructive hypothesis:**

- Melanocytes are destroyed by toxic substances formed as part of normal melanin biosynthesis, e.g. dopa and dopachrome.
- Inhibition of thioredoxin reductase a free radical scavenger on the membrane of melanocytes, it is inhibited by calcium; so high extracellular Ca causes increased superoxide radicals leading to inhibition of tyrosinase by upsetting equilibrium of oxidized and reduced thioredoxin in epidermis.

4. **Neural theory:**

- Neurochemical mediators from the nearby nerve endings would be involved in the destruction of melanocytes. This provides an explanation for segmental vitiligo.
- There is increased sweating and vasoconstriction in the affected area that may be due to increased adrenergic activity.

q 9

* Ocular manifestation in both (OCA, OA)
- reduction of melanin pigment within eye structures → Translucent iris that transmits light upon globe trans illumination
→ relatively hypopigmented retina, fovea leading to:
* photophobia ,
* decreased visual acuity.
* severity of the findings correlates with the amount of reduction of melanin pigment

- mis routing of optic nerve fibres during development →
* strabismus
* Nystagmus
* Binocular vision loss

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Pigmentary:

Q10 DD of acquired hypomelanosis?

1. Vitiligo.
   (Acquired destruction of melanocytes).
2. Vogt-Koyanagi-Harada syndrome.
   (Due to aberrent migration or survival of melanocytes).
3. Chemical or drug-induced leukoderma.
4. Post-inflammatory hypo/de-pigmentation.
5. Pityriasis Versicolor.
6. Cutaneous infection (e.g. leprosy).
7. Treatment e` potent topical corticosteroid may lead to hypomelanosis.